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Original article

Combined mechanical mitral valve replacement and transmitral myectomy for hypertrophic obstructive cardiomyopathy treatment: An experience of over 20 years

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ABSTRACT

Background: Although transaortic septal myectomy (TASM) is recognized as a standard procedure for treating hypertrophic obstructive cardiomyopathy (HOCM), occasionally the left ventricle (LV) intracavitary gradient remains postoperatively because of this technically demanding procedure. Mitral valve replacement (MVR) is sometimes chosen as an alternative option, but data on its long-term outcomes are lacking.
Methods and results: Between 1991 and 2016, 29 patients [age, 14–82 (mean 58.9 ± 15.9) years; 22 female patients (75.9%)] underwent combined mechanical MVR and transmitral myectomy. Of these, six patients had undergone MVR following a second cardiac arrest because of the residual LV outflow gradient or residual mitral regurgitation following TASM. Concomitant TASM was performed in 13 patients. The LV intracavitary gradient at rest assessed by transthoracic echocardiography significantly decreased postoperatively (16.8 ± 19.1 mmHg vs. 107.4 ± 52.5 mmHg, $p < 0.0001$). Actuarial freedom rates from cardiac death were 92.8%, 89.0%, and 80.1% at 5, 10, and 15 years postoperatively, respectively. Sudden death occurred in three of the four patients who died of late cardiac complications. None of these patients with sudden death had implantable cardioverter-defibrillators. Most patients had maintained their LV end-diastolic dimension at <50 mm for 10–15 years postoperatively. Actuarial freedom rates from hospitalization for heart failure were 87.7%, 82.2%, and 54.8% at 5, 10, and 15 years postoperatively, respectively. Occurrence rates of cerebral hemorrhage and infarction were 0.6% per patient-year and 1.3% per patient-year, respectively.
Conclusions: Combined mechanical MVR and myectomy is an effective procedure to eliminate the LV intracavitary gradient in patients with HOCM. Although this procedure remains a viable option in certain situations, optimal medical treatment and close clinical follow-up along with the cooperation between cardiac surgeons and cardiologists are necessary to achieve favorable long-term outcomes.

Introduction

Intraventricular obstruction in the left ventricle (LV) in hypertrophic cardiomyopathy (HCM) leads to increased intracavitary pressure and LV wall stress, prolonged LV relaxation, decreased LV filling and cardiac output, myocardial ischemia, secondary mitral regurgitation (MR), and ventricular arrhythmias [1,2]. Maron et al. [3] have reported that 37% of patients with HCM developed an LV outflow gradient ≥50 mmHg at rest and 23% of the patients developed a dynamic LV outflow gradient ≥50 mmHg during exercise. There are several causes of the LV cavitary obstruction, including a protrusion of the hypertrophied septum into the LV cavity, systolic anterior motion (SAM) of the mitral

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valve [4], direct insertion of the anomalous papillary muscle into the middle of the anterior mitral leaflet, and thickened secondary chords [2,5]. SAM of the mitral valve is the prevalent pathophysiological component responsible for the dynamic obstruction and MR during systole in patients with HCM. The mechanism underlying SAM could potentially be explained by the displacement of the papillary muscle and elongation of the mitral valve apparatus, thereby causing the mitral leaflets to interpose into the LV outflow stream of the blood flow, which in turn, exerts drag forces on the anterior component of the mitral valve, pulling the mitral leaflets up into the LV outflow tract [2,6,7].

Transaortic septal myectomy (TASM) represents the standard procedure for hypertrophic obstructive cardiomyopathy (HOCM) with favorable long-term results as reported by several high-volume centers [8–11]. However, TASM is a technically demanding procedure that requires an adequate resection deep to the base of papillary muscles using an appropriate width and depth through a limited available surgical field [12,13]. Furthermore, HOCM often involves abnormalities of the mitral valve including excess length of the anterior and posterior mitral leaflet or a restricted mitral leaflet [14,15]. Various repair techniques for mitral valve and subvalvular apparatus have been implemented to manage these abnormalities [14,16–19].

Mitral valve replacement (MVR) has been performed as an option for the surgical treatment of HOCM [20–22]. MVR is a reliable procedure used to obliterate the LV intracavitary gradient because of SAM. During MVR, papillary muscles and abnormal muscle bundles can be simultaneously resected through the mitral orifice to augment the LV cavity (transmitral myectomy). The mechanical mitral valve has been commonly used because its profile is significantly lower than that of a bioprosthetic mitral valve. However, MVR with a mechanical valve presents inherent risks of anticoagulant complications, thromboembolism, and valve dysfunction. Furthermore, long-term results of combined mechanical MVR and myectomy for patients with HOCM remain unknown. This study evaluates the postoperative outcomes of patients with HOCM following this procedure.

Material and methods

Between January 1991 and December 2016, 48 patients with HOCM underwent surgical treatment including TASM with or without mitral repair, transapical myectomy, and MVR with myectomy. Of these patients, 24 patients (50.0%) underwent combined MVR with transmitral myectomy and TASM or MVR with transmitral myectomy as scheduled (mechanical MVR: 23 patients, bioprosthetic MVR: 1 patient). Although 22 patients (45.8%) underwent TASM, 7 patients eventually underwent conversion to MVR with transmitral myectomy (mechanical MVR: 6 patients, bioprosthetic MVR: 1 patient). Consequently, 29 patients who underwent mechanical MVR with myectomy were enrolled in this study (Fig. 1).

Informed consent for this study was waived because of the retrospective and observational study design.

Diagnosis of HOCM

HOCM was diagnosed on the basis of the following criteria: (1) LV thickness of ≥15 mm in one or more segments, as assessed by transthoracic echocardiography; (2) resting or provoked LV intracavitary gradient of ≥30 mmHg, as measured by continuous wave Doppler assessment; (3) no moderate or severe aortic stenosis; and (4) no diseases similar to HCM that present with LV hypertrophy such as Fabry disease, glycogen storage disease, mitochondrial disease, Noonan syndrome, or mucopolysaccharidosis.

Indication for surgical treatment

Surgical treatment was performed in patients whose clinical symptoms did not improve despite the optimal medical treatment and whose LV intracavitary gradient was ≥50 mmHg at rest or during physiologic provocation.

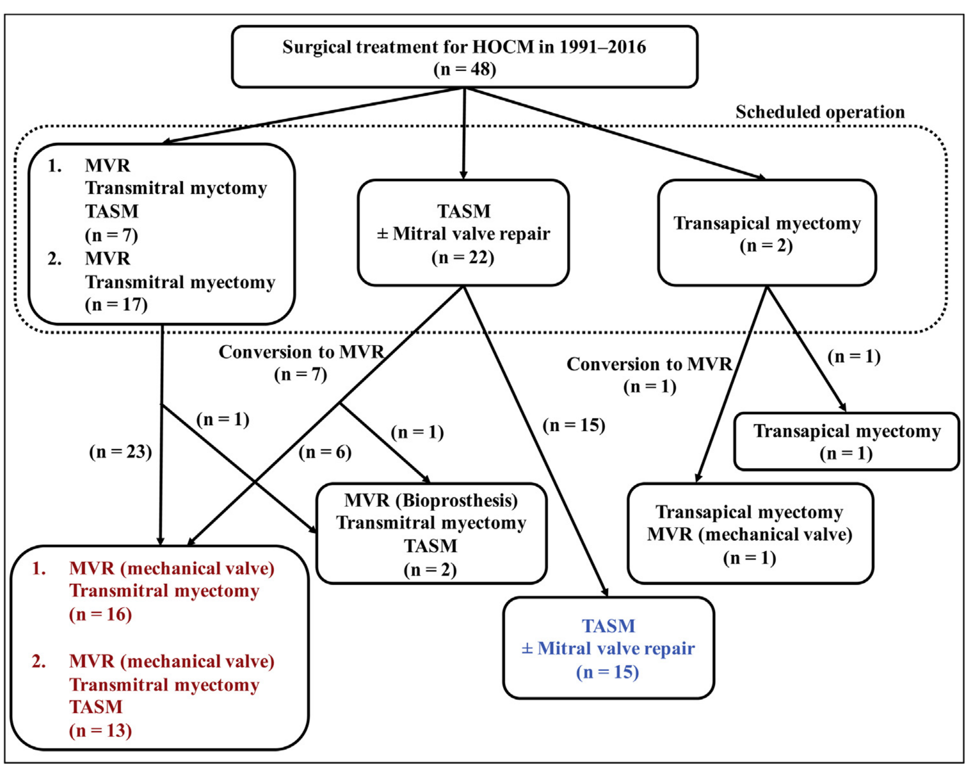

Fig. 1. Inclusion and exclusion criteria for patient selection and study protocol. *Abbreviations*: HOCM, hypertrophic obstructive cardiomyopathy; MVR, mitral valve replacement; TASM, transaortic septal myectomy.

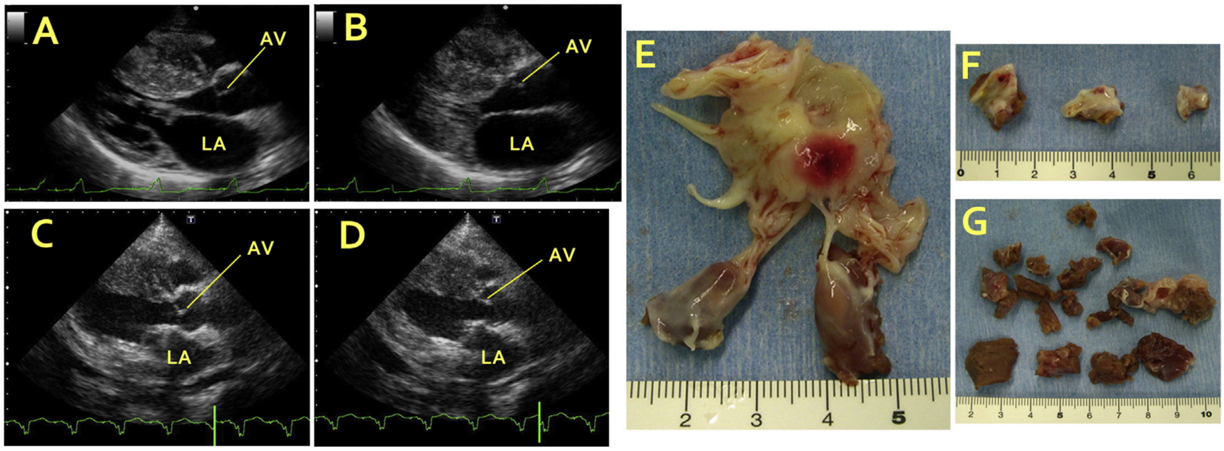

Fig. 2. Pre- and postoperative transthoracic echocardiography and intraoperatively excised specimen in a 44-year-old female patient with hypertrophic obstructive cardiomyopathy who underwent combined mechanical mitral valve replacement with transmitral myectomy and transaortic septal myectomy. (A, B) Preoperative transthoracic echocardiography image at a diastolic and a systolic phase (LVDd: 33 mm, LVDs: 17 mm, LVFS: 49%, septal wall thickness: 35 mm, posterior wall thickness: 10 mm). Diffusely hypertrophied septum and thickened secondary chord are detected. The LV outflow cavity is obstructed by them at the systolic phase. (C, D) Early postoperative transthoracic echocardiography image at a diastolic and a systolic phase (LVDd: 35 mm, LVDs: 25 mm, LVFS: 29%, septal wall thickness: 17 mm, posterior wall thickness: 6 mm). LV cavity is significantly augmented by the resection of papillary muscles and abnormal muscle bundles. There is no LV intracavitary gradient. (E) Resected anterior mitral leaflet and papillary muscles. (F) The excised subaortic septal muscles. These muscles were resected through the aortic valve. (G) Excised papillary muscles and abnormal muscle bundles. These muscles were resected through the mitral orifice (transmitral myectomy). *Abbreviations*: AV, aortic valve; LA, left atrium; LVDd, left ventricle end-diastolic dimension; LVDs, left ventricle end-systolic dimension; LVFS, left ventricle fractional shortening.

Surgical strategy for HOCM

Until 2008, we had frequently performed MVR with transmitral myectomy in patients with severe HOCM who had a diffuse LV hypertrophy, because it is a reliable procedure to eliminate the LV intracavitary gradient and to enlarge the LV cavity. TASM had been concomitantly performed in patients with significant subaortic septal hypertrophy. Starting in 2009, TASM with or without mitral repair was performed for HOCM patients with a diffuse LV hypertrophy as a standard procedure, based on the experiences acquired over the years, to avoid complications of artificial heart valves by preserving the native mitral valve apparatus. In cases where TASM had failed, conversion to MVR with transmitral myectomy was performed intraoperatively. Recently, transapical myectomy became an option of choice in midventricular HOCM patients afflicted by low output syndrome or ventricular arrhythmias [23,24].

Operative techniques

Following general anesthesia, a transesophageal echocardiography probe was inserted into the esophagus to examine the pre- and postoperative cardiac anatomy and assess hemodynamic performance. Cardiac arrest was used under standard cardiopulmonary bypass with moderate hypothermia and tepid blood cardioplegia via median sternotomy. The right-sided left atrium was opened and both the anterior and posterior mitral leaflets were resected including all papillary muscles and abnormal muscle bundles around papillary muscles through the mitral orifice (transmitral myectomy). Then, the mitral mechanical valve was implanted using everting mattress suturing technique with 2-0 braided polyester sutures. All mechanical mitral valves were bileaflet. TASM was performed according to Morrow's operative technique [25] before MVR in a situation of subaortic septal hypertrophy (Fig. 2).

In patients who underwent TASM with or without mitral repair, when a significantly residual LV intracavitary gradient or MR remained following TASM assessed by an intraoperative transesophageal echocardiography or a pressure measurement using a needle inserted into the LV cavity, subsequent MVR with transmitral myectomy was performed under a second cardiac arrest. Concomitant procedures including coronary artery bypass grafting, aortic valve replacement (AVR) with mechanical bileaflet valve, cryomaze procedure, or tricuspid annuloplasty were performed as needed depending on the patient's comorbidities.

Postoperative follow-up

Patients received warfarin sodium with monitoring of the target international normalized ratio (INR) of 2.0–3.0. Patients who underwent concomitant coronary artery bypass grafting additionally received an antiplatelet agent (aspirin 100 mg). All patients were monitored at our center or at other outpatient clinics. Telephone or questionnaire-based follow-up was performed in patients who did not visit our center within the previous year of data collection.

Statistics

Data are expressed as means ± standard deviation, frequencies (percentages), or as medians (interquartile ranges). The normality of the distribution of continuous variables was assessed using the Shapiro–Wilk test. Between-group differences in parametric and non-parametric variables were assessed using Student *t*-test and Mann–Whitney *U*-test, respectively; between-group differences in categorical variables were assessed using a Pearson chi-squared test or Fisher exact test. Probability-values (*p*-value) of <0.05 were considered statistically significant. Actuarial survival and freedom from adverse events were calculated according to the Kaplan–Meier method. Statistical analyses were performed using the JMP 11 software (SAS Institute Inc., Cary, NC, USA).

Results

Preoperative patient characteristics are summarized in Table 1. Patient age ranged from 14 to 82 (mean: 58.9 ± 15.9) years, and there were 22 female patients (75.9%). Patients who underwent MVR with transmitral myectomy had a higher rate of HCM family history (34.5% vs. 6.7%, $p = 0.0289$) and severe New York Heart Association functional class (≥III) (72.4% vs. 0%,

Table 1
Preoperative patient characteristics (comparison with transaortic septal myectomy).

Variables	MVR + transmitral myectomy ± TASM	TASM ± mitral repair	*p*-Value
n	29	15	
Age (years)	58.9 ± 15.9	63.2 ± 12.1	0.3772
Female	22 (75.9%)	7 (47.7%)	0.0550
NYHA functional class			
I	0	0	
II	8 (27.6%)	15 (100%)	<0.0001
III	20 (69.0%)	0	<0.0001
IV	1 (3.4%)	0	0.3578
Family history of HCM	10 (34.5%)	1 (6.7%)	0.0289
History of VT/Vf	4 (13.8%)	2 (13.3%)	0.9663
Atrial fibrillation	2 (6.9%)	1 (6.7%)	0.9771
Pacemaker implantation	1 (3.4%)	0	0.3578
ICD implantation	1 (3.4%)	0	0.3578
History of PTSMA	2 (6.9%)	2 (13.3%)	0.4919
Cre > 1.5 mg/dl	1 (3.4%)	1 (6.7%)	0.6358
Active IE	2 (6.9%)	0	0.1901
Disease duration (years)	5.5 ± 5.5	6.5 ± 5.0	0.5470

Abbreviations: Cre, serum creatinine; HCM, hypertrophic cardiomyopathy; ICD, implantable cardioverter-defibrillators; IE, infectious endocarditis; NYHA, New York Heart Association; MVR, mitral valve replacement; PTSMA, percutaneous transluminal septal myocardial ablation; TASM, transaortic septal myectomy; Vf, ventricular fibrillation; VT, ventricular tachycardia.

$p < 0.0001$) compared with those who underwent TASM. The mean disease duration after the development of symptoms because of HOCM in MVR with transmitral myectomy was similar in TASM (5.5 ± 5.5 years vs. 6.5 ± 5.0 years, $p = 0.5470$).

Preoperative transthoracic echocardiography data are shown in Table 2. Patients who underwent MVR with transmitral myectomy had a higher septal wall thickness than those who underwent TASM (23.9 ± 6.3 mm vs. 19.6 ± 5.1 mm, $p = 0.0411$). In addition, the rate of patients who had ≥20 mm of septal wall thickness is higher in MVR with transmitral myectomy than in TASM (69.0% vs. 26.7%, $p = 0.0069$). The rate of patients who had a contact between the posterior wall or the papillary muscle and septal wall in the LV mid-portion is also higher in MVR with transmitral myectomy than in TASM (79.3% vs. 40.0%, $p = 0.0096$).

Seven patients underwent mechanical MVR with transmitral myectomy concomitantly with TASM as scheduled. By contrast, there were 7 out of 22 patients who underwent scheduled TASM received conversion to MVR (mechanical valve: $n = 6$, bioprosthetic valve: $n = 1$) following a second cardiac arrest because of the residual LV outflow gradient or residual MR after TASM. These occurred precisely because resection of the LV muscle was insufficient and caused MR because of residual SAM, with the exception in one patient who had residual MR due to unsuccessful repair for bileaflet prolapse. Taken together, a total of 13 patients underwent combined mechanical MVR with transmitral myectomy and TASM (Fig. 1).

AVR with mechanical valve was performed as a concomitant procedure in three patients with aortic regurgitation (AR), among which two had active infectious endocarditis. Other concomitant procedures included coronary artery bypass grafting (four patients), cryomaze procedure (one patient), and tricuspid annuloplasty (one patient). Two intraoperative complications occurred, and were repaired during the operation, including rupture of the LV free wall after MVR and aortic valve injury during

Table 2
Preoperative transthoracic echocardiography data (comparison with transaortic septal myectomy).

Variables	MVR + transmitral myectomy ± TASM	TASM ± mitral repair	*p*-Value
Intra-LV cavity PG at rest (mmHg)	107.4 ± 52.5	81.0 ± 48.9	0.1210
Intra-LV cavity PG at rest ≥30 mmHg	28 (96.6%)	14 (93.3%)	0.6358
Septal wall thickness (mm)	23.9 ± 6.3	19.6 ± 5.1	0.0411
Septal wall thickness ≥20 mm	20 (69.0%)	4 (26.7%)	0.0069
Septal wall thickness ≥30 mm	6 (20.7%)	1 (6.7%)	0.2002
LV posterior wall thickness (mm)	14.4 ± 4.6	15.0 ± 5.1	0.7026
LV end-diastolic dimension (mm)	40.8 ± 5.9	43.5 ± 5.1	0.0778
LV end-systolic dimension (mm)	21.8 ± 6.0	25.3 ± 4.6	0.0540
LV fractional shortening (%)	46.5 ± 9.7	41.7 ± 8.8	0.1140
MR grade			
≤Trivial	6 (20.7%)	2 (13.3%)	0.5404
Mild	5 (17.2%)	7 (46.7%)	0.0411
Moderate	14 (48.3%)	6 (40.0%)	0.6004
Severe	4 (13.8%)	0	0.0599
SAM	22 (75.9%)	15 (100%)	0.0108
Mitral leaflet prolapse	3 (10.3%)	1 (6.7%)	0.6805
Contact between PM/PW and septal wall in LV mid-portion	23 (79.3%)	6 (40.0%)	0.0096
Direct PM insertion to AML or thickened secondary chord	7 (24.1%)	5 (33.3%)	0.5200
Mitral stenosis ≥ moderate	2 (6.9%)	0	0.1901
AR ≥ moderate	2 (6.9%)	1 (6.7%)	0.9771
TR ≥ moderate	2 (6.9%)	1 (6.7%)	0.9771

Abbreviations: AML, anterior mitral leaflet; AR, aortic regurgitation; LV, left ventricle; MR, mitral regurgitation; MVR, mitral valve replacement; PG, pressure gradient; PM, papillary muscle; PW, posterior wall; SAM, systolic anterior motion; TASM, transaortic septal myectomy; TR, tricuspid regurgitation.

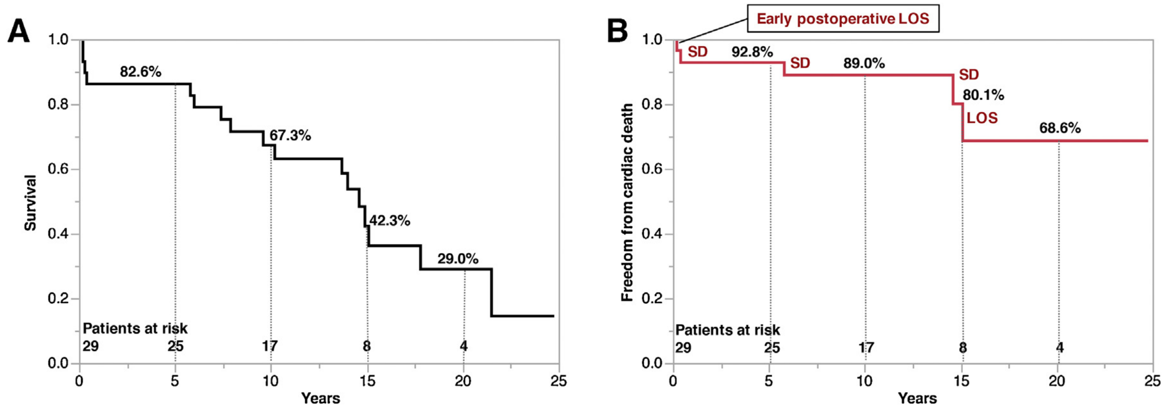

Fig. 3. Survival and freedom from cardiac death. (A) Survival. (B) Freedom from cardiac death. Causes of death have been indicated lateral to the curve. *Abbreviations*: LOS, low output syndrome; SD, sudden death.

TASM. A new pacemaker implantation was performed early after operation in two patients (6.9%) who developed a complete atrioventricular block postoperatively.

Postoperative transthoracic echocardiography showed that the LV cavitary gradients at rest significantly decreased postoperatively (16.8 ± 19.1 mmHg vs. 107.4 ± 52.5 mmHg, $p < 0.0001$). No patient presented with an LV outflow gradient of $\geq$20 mmHg postoperatively. However, in seven patients (24.1%), the LV intracavitary gradient of $\geq$30 mmHg remained at the midventricular portion between residual papillary muscles and the septum. A contact between the mid-portion of the septum and residual papillary muscles or the posterior LV wall during systole remained in 10 patients (34.5%) postoperatively.

Two patients (6.9%) died of early postoperative low output syndrome (71 days postoperatively) and acute exacerbation of interstitial pneumonia (107 days postoperatively) in the hospital without being discharged. The former patient had undergone MVR with TASM and cryomaze procedure in 1993, and developed decreased LV function with severe paravalvular leak of mechanical mitral valve just after the operation. This patient required prolonged catecholamine and respiratory support, and finally died without a redo operation. Actuarial survival rates were 82.6%, 67.3%, and 42.3% at 5, 10, and 15 years postoperatively, respectively (Fig. 3A). Actuarial freedom rates from cardiac death were 92.8%, 89.0%, and 80.1% at 5, 10, and 15 years postoperatively, respectively (Fig. 3B). Sudden death accounted for three-fourths of all postoperative cardiac deaths in patients who survived the operation and of which none had an implantable cardioverter-defibrillator. The mean follow-up period was 11.0 ± 6.7 years with a collective follow-up of 319.3 patient-years after operation. None of the patients were lost to follow-up.

Changes in LV end-diastolic dimension (LVDd) and LV fractional shortening (LVFS) during the follow-up period are shown in Fig. 4. In many patients, LVDd and LVFS decreased during the early postoperative period compared with those during the preoperative period. However, LVDd increased to almost the same level as preoperative values in 1–5 years following the operation, and LVDd was maintained at <50 mm for the following 10–15 years. LVFS was maintained at >30% in most of the patients. However, in one patient, LVDd markedly increased with a significantly decreased LVFS over 15 years following the operation: 70 mm of LVDd and 4% of LVFS at 20 years postoperatively.

Changes in the septal wall thickness and the posterior wall thickness are presented in supplementary Fig. 1. The septal wall thickness decreased early postoperatively and was maintained at a level of about 11–18 mm during the follow-up period. The posterior wall thickness decreased early postoperatively in patients who had >15 mm of the wall thickness, and it was maintained at the level of about 10–16 mm during the follow-up period.

Actuarial freedom rates from permanent atrial fibrillation (AF) were 69.2%, 69.2%, and 46.7% at 5, 10, and 15 years, postoperatively, respectively (Fig. 5A). Actuarial freedom rates from hospitalization for heart failure (first hospitalization for heart failure postoperatively in each patient) were 87.7%, 82.2%, and 54.8% of patients at 5, 10, and 15 years postoperatively, respectively (Fig. 5B). Causes of heart failure were mainly related to AF and renal insufficiency (serum creatinine > 1.5 mg/dL). The patient who was hospitalized for AR at 6 months following the combined MVR, transmitral myectomy, and TASM developed moderate AR during the early postoperative period (Table 2) and underwent AVR with a mechanical valve for severe AR 3 years postoperatively. A patient who developed severely dilated LV cavity with decreased LV contractility was hospitalized 16 years postoperatively.

Occurrence rates of cerebral hemorrhage and infarction were 0.6% per patient-year and 1.3% per patient-year, respectively. Actuarial freedom rates from a cerebral hemorrhage were 94.1% and 86.7% of patients, and from infarction were 95.8% and 81.1% of patients at 10 and 15 years postoperatively, respectively (Fig. 6).

Discussion

TASM is perceived as a standard procedure for HOCM patients and Hong et al. [26] mentioned that MR related to SAM resolves after adequate septal myectomy, and concomitant mitral valve surgery is rarely required unless the patient has intrinsic mitral disease. However, in the present study, MVR was performed in a half of the patients for the treatment of HOCM. We had frequently chosen MVR with transmitral myectomy in patients with severe HOCM until 2008, because it is a reliable procedure to eliminate outflow tract obstruction and to augment LV cavity. In addition, six patients (27.3%) had undergone a conversion from TASM to mechanical MVR and one patient (4.5%) a conversion from TASM to bioprosthetic MVR. The most common reason for the conversion was insufficient resection of the LV muscle. While we performed TASM in 11 patients since 2009, two patients had undergone a conversion from TASM to MVR. TASM requires that the surgeon has gained adequate experience, despite the low number of patients who require surgical treatment for HOCM, thus TASM is perceived as a more technically demanding procedure. The more diffuse the LV hypertrophy becomes, the more difficult the TASM procedure becomes to achieve sufficient muscle resection. The annual report by the Japanese Association for Thoracic Surgery in 2014 [27] showed only 171 cases of myectomy for HOCM, including

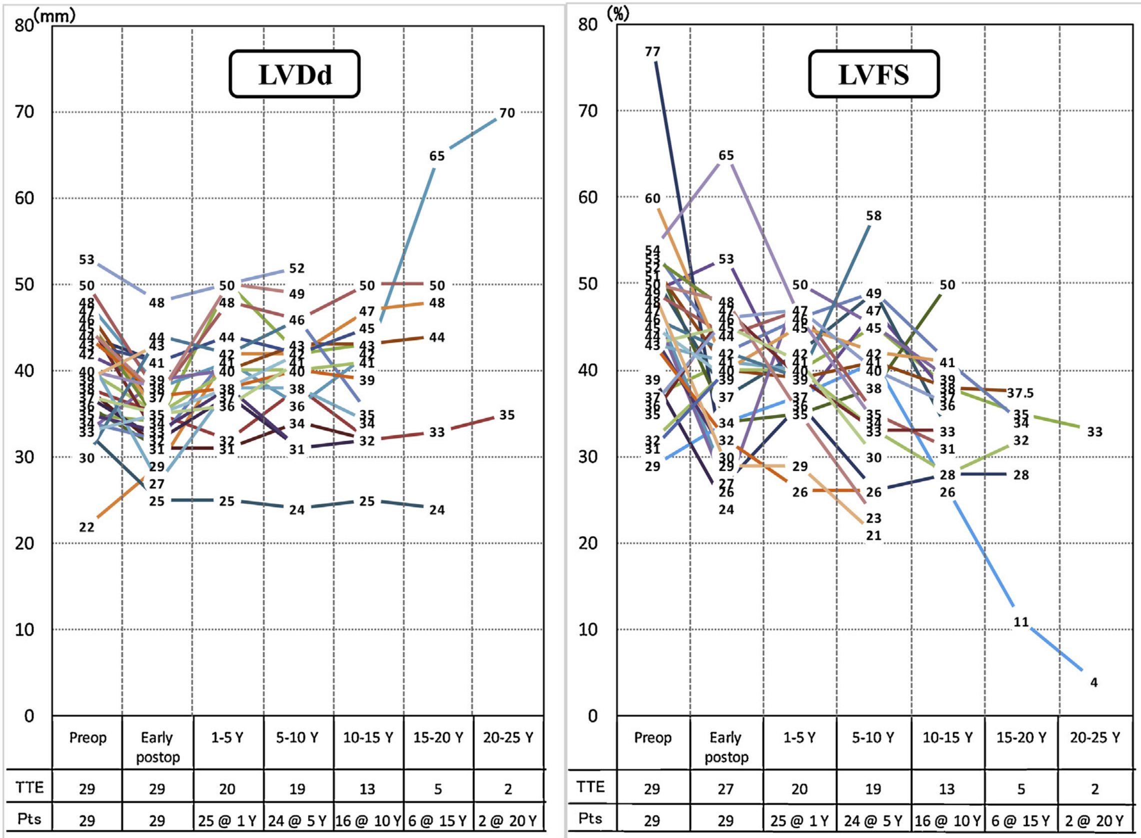

Fig. 4. Changes in left ventricle end-diastolic dimension and left ventricle fractional shortening during the follow-up period. *Abbreviations*: LVFS, left ventricle fractional shortening; Pts, the number of patients in each period; TTE, the number of patients who underwent transthoracic echocardiography in each period; Y, years.

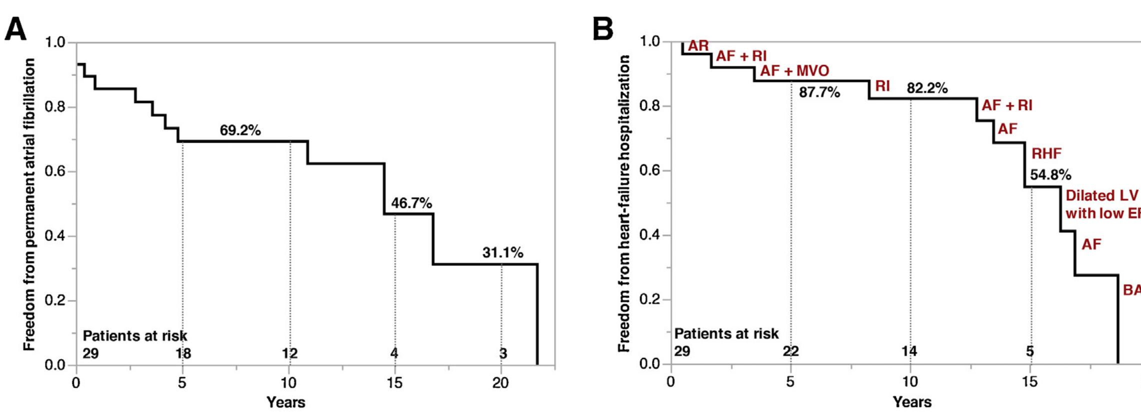

Fig. 5. Freedom from permanent atrial fibrillation and hospitalization due to heart failure. (A) Freedom from permanent atrial fibrillation. (B) Freedom from hospitalization due to heart failure. Comorbidities which were associated with hospitalization due to heart failure are indicated lateral to the curve. *Abbreviations*: AF, atrial fibrillation; AR, aortic regurgitation; BA, bronchial asthma; EF, ejection fraction; LV, left ventricle; MVO, midventricular obstruction in left ventricle; RHF, right heart failure; RI, renal insufficiency (serum creatinine > 1.5 mg/dL).

110 concomitant cases of AVR with localized subaortic septal bulge. Given the particularity of HOCM and TASM being a technically demanding procedure, combined MVR and myectomy remains an alternative option.

MVR with a mechanical valve is a well-established and simple procedure to eliminate the LV outflow gradient because of SAM. However, in our result, although there were no patients who had a residual LV intracavitary gradient at the outflow tract postoperatively, there were seven patients (24.1%) who had an LV intracavitary gradient of ≥30 mmHg within the midventricular portion between residual papillary muscles and the septum. As papillary muscles can be resected to their origin from the mitral orifice with relative ease, all papillary muscles and surrounding abnormal muscle bundles should be thoroughly resected. Residual midventricular obstruction may increase the risk of heart failure and ventricular arrhythmias later for the patient.

Combined mechanical MVR and myectomy not only eliminates the LV intracavitary gradient, but it may also improve the LV compliance. Said et al. [23] have reported that an augmented LV after myectomy contributed to a larger LV volume during end-diastole, a lower end-diastolic LV pressure, and an increased stroke volume calculated by contrast ventriculograms and pressure

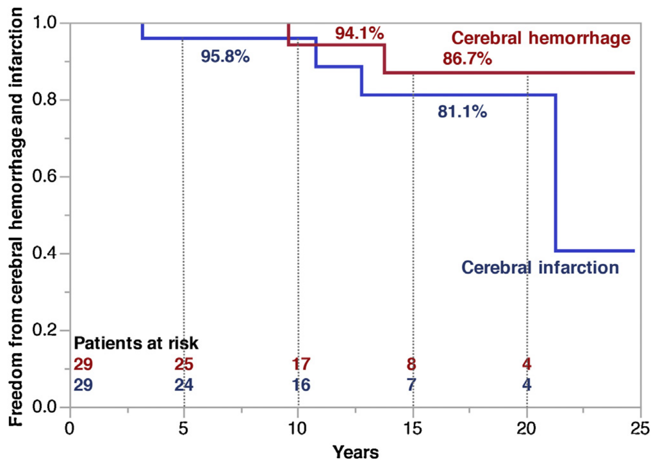

Fig. 6. Freedom from cerebral hemorrhage and infarction.

measurements. However, concerns remain regarding changes in LV size and contractility after MVR with myectomy. In this study, LVDd and LVFS decreased during the early postoperative period and it cannot be excluded that LV compliance temporally decreased because of the operation. After that, although LVDd increased at 1–5 years postoperatively and both LVDd and LVFS were maintained at an almost normal level in most of the patients during the follow-up period, except for one patient who developed a severely dilated LV with reduced LV contractility over 15 years following the operation. Harris et al. [28] have reported that the timeframe between the onset of HCM symptoms and the end-stage phase of HCM was 14 ± 10 years, and the interval from end-stage phase to death or heart transplantation was 2.7 ± 2 years. The authors mentioned that the most frequent pattern of LV remodeling was LV cavity enlargement and/or a progressively increasing LV cavity. In this study, the mean interval from the onset of HOCM symptoms to surgery was 5.5 ± 5.5 years. Consequently, patients who underwent combined mechanical MVR and myectomy will avoid significantly increasing LV cavity within 15–20 years after the onset of HOCM symptoms, which suggests that the procedure does not accelerate the progression of LV remodeling.

Risk factors for sudden cardiac death (SCD) after MVR with myectomy are unknown, although SCD with HCM is associated in patients with a family history of SCD, a maximal LV wall thickness of >30 mm, syncope, abnormal blood pressure response during exercise, and non-sustained ventricular tachycardia [8,9]. In this study, sudden death occurred in three of the four patients who died of late cardiac complications, and none of these patients had an implantable cardioverter-defibrillator. A risk stratification protocol of SCD in patients with HOCM having undergone surgery may need to be established to help making a decision regarding the use of implantable cardioverter-defibrillator postoperatively [29]. To achieve this goal, a close follow-up of patients, an assessment of the severity of disease [30], and the guidance of both cardiac surgeons and cardiologists are necessary.

AF is the most common form of arrhythmia and is associated with high thromboembolic events in patients with HCM. Guttmann et al. [31] have reported that the overall incidence of AF in patients with HCM was 3.8% per year and that the incidence of thromboembolism in these patients was 3.75% per year. Anticoagulation therapy using a vitamin K antagonist is recommended in patients with HCM who developed paroxysmal AF [9,10]. In this study, approximately 25% of patients developed permanent AF at 5 years postoperatively and development of AF was frequently associated with hospitalization for heart failure. Thus, combined mechanical MVR and myectomy for HOCM does not attenuate the development of AF. Further, occurrence rates of cerebral infarction and hemorrhage in our patients were slightly higher than those reported in patients with mitral stenosis or mitral regurgitation who had undergone mechanical MVR (cerebral infarction, 1.3% vs. 0.2–0.8% per patient-year; cerebral hemorrhage, 0.6% vs. 0.2% per patient-year) [32,33]. Optimal postoperative medical treatment is crucial for the prevention and/or control of AF and anticoagulant therapy-related complications.

Although mitral bioprosthetic valves prevent complications associated with anticoagulation therapy and valve-related thromboembolic events, they present a higher profile and a lower durability than mechanical valves. However, some recent mitral bioprosthetic valves have a lower profile and protrude into the LV outflow tract to a lesser extent than previous generations of mitral bioprosthetic valves [34]. The use of these bioprosthetic valves is an alternative option for patients who have adequately augmented LV outflow tract. This is particularly true in female patients with childbearing potential, as prosthetic valve selection is a serious issue in such cases [35]. Therefore, sufficient explanation for the probable requirement of MVR, prosthetic valve selection, and long-term planning are essential in patients with HOCM before the surgery.

This study has certain limitations. This is a single-center retrospective observational study with a small number of patients. Various types and sizes of bileaflet mechanical valves were used for MVR. Because monitoring of INR targets was performed at our center or at other outpatient clinics, control of INR may have varied between hospitals. Further, we did not compare postoperative results of MVR with transmitral myectomy to those of TASM for the following reasons. First, there were the small number of patients who underwent TASM. Second, the severity of HOCM was different between these two groups; patients with MVR had more serious HOCM with extremely small LV cavity. Third, many patients with TASM had significantly shorter follow-up periods than those with MVR.

Conclusions

Combined mechanical MVR and myectomy is an effective procedure to eliminate the LV intracavitary gradient. This procedure remains a viable option in patients with TASM failure, severely diffuse LV hypertrophy, and at low risk of adverse events related to anticoagulation therapy. However, pre- and postoperative optimal medical treatment and a close clinical follow-up monitored in cooperation between cardiac surgeons and cardiologists are necessary to achieve favorable long-term outcomes by preventing SCD, mechanical valve-related events, and heart failure.

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Conflict of interest

All authors declare that there is no conflict of interest.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.jjcc.2018.12.012.

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